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Originally published at:
Gerspach, Christian; Wirz, Mirjam; Knubben-Schweizer, Gabriela; Braun, Ueli (2011). Thrombosis of the cranial vena cava in a cow with bronchopneumonia and traumatic reticuloperitonitis. Canadian Veterinary Journal, 52(11):1228-1231.

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C. Gerspach, M. Wirz, G. Schweizer-Knubben, U. Braun

C. Gerspach, DrMedVet., **G. Schweizer-Knubben**, DrMedVet., **U. Braun**, DrMedVet., Department of Farm Animals, **M. Wirz**, MedVet, Institute of Veterinary Pathology, University of Zurich, Winterthurerstrasse 260, CH-8057 Zurich

Abstract

This paper reports the clinical findings, surgical and medical management, and necropsy of a 6 year old cow with thrombosis of the cranial vena cava and thrombo-embolic pneumonia following traumatic reticuloperitonitis. The clinical diagnosis was confirmed by necropsy.

Thrombosis of the caudal vena cava is a well known disorder of cattle, whereas thrombosis of the cranial vena cava is relatively uncommon (1-7). Liver abscesses that break into the caudal vena cava are the most common cause of thrombosis of the vessel (3,8). Thrombosis of the cranial vena cava is usually attributable to embolism of a jugular vein thrombus and is less often due to haematogenous spread of infection (6). Thrombosis of the vena cava is often associated with metastatic bronchopneumonia with characteristic clinical signs. Wyssmann (1), Breeze (9) and Bueno (10) described respiratory syndrome and signs of congestion in cattle with thrombosis of the cranial vena cava.

This case report describes the clinical findings in a six-year-old Swiss Braunvieh cow with thrombosis of the cranial vena cava.

Case description

The cow had calved three months ago. Three weeks ago she had a reduced appetite and was treated by the referring veterinarian with a magnet and procaine penicillin because of suspected traumatic reticuloperitonitis. There was a transient response to treatment, but two weeks later milk production decreased and rumination ceased. She was then referred to the Department of Farm Animals, University of Zurich, for further diagnostic work-up.

The general condition and mental status of the cow were markedly abnormal, the cow was depressed. The cow had a body condition score of 3/5. The rectal temperature was 38.7 °C, and the heart rate was 108 bpm. Both jugular veins were distended and there was brisket edema (Fig. 1). The respiratory rate was 48 breaths/minute, and the cow had abdominal breathing and coughed spontaneously. Auscultation of the lungs revealed increased breath sounds. Ruminal motility was slightly decreased with two contractions per three minutes, and there was a reduced amount of ruminal content. The withers' pinch, pole test and deep palpation of the cranio-ventral abdomen were consistently positive, and the glutaraldehyde test was shorter than normal at 3 minutes (normal > 10 minutes).

Further diagnostic work-up included haematological and biochemical analyses and ultrasonographic examination of the lungs (11), heart (12), reticulum (13), liver (14) and abdomen (15). Radiographs of the lungs and reticulum were also taken (16).

The most important haematological and biochemical findings were an increase in the concentrations of total protein at 84 g/l (normal 60-80 g/l) and fibrinogen at 8 g/l (normal 4-7 g/l) and increased activities of glutamate dehydrogenase at 39.1 U/l (normal 0-25 U/l), sorbit dehydrogenase at 37.2 U/l (normal 0-20 U/l) and γ-glutamyl transferase at 41 U/l (normal 0-20 U/l). The albumin concentration was normal (29 g/l; normal 21-36 g/l).

Ultrasonographic examination of the reticulum (13) showed one weak incomplete contraction per 3-minute period (normal, 3 complete biphasic contractions per 3-minute period). Echogenic fibrin deposits containing pockets of hypoechogenic fluid were seen on the reticular wall and extended from the left to right side of the abdomen and from the ventral abdomen to the level of the elbows. An 11-cm abscess with a hypoechogenic centre surrounded by an echogenic capsule was

seen caudal to the reticulum. Fibrin deposits were seen on the ventral margin of the liver. The pleura had comet-tail artefacts on both sides of the thorax, and a layer of anechoic fluid with a diameter of 1 cm was seen between the visceral and parietal pleura on the right side. The heart valves, liver parenchyma, caudal vena cava, portal vein and gallbladder were unremarkable. Transcutaneous, ultrasound-guided biopsy of the liver was done because of the elevation in liver enzyme activity; histological evaluation of the sample did not reveal pathologic changes.

On laterolateral radiographs of the thorax the lungs, caudal cardiac silhouette and caudal vena cava were clearly seen. A non-delineated soft-tissue opacity was seen in the caudoventral lung field, and the dorsal lung area appeared normal. This finding was interpreted as localised pneumonia. Radiographs of the reticulum revealed two magnets on the ventral aspect of the reticulum. One magnet had two linear foreign bodies, one of which appeared to be at an angle to the magnet and possibly penetrating the reticular wall. There was localised loss of detail in the region of the caudal reticular contour.

Traumatic reticuloperitonitis and bronchopneumonia were diagnosed based on all the findings. The cause of distension of both jugular veins was suspected to be obstruction of the cranial vena cava by a thrombus or compression of the vein by a mass. Cardiac insufficiency was ruled out based on echocardiography.

The albumin concentration was normal, therefore a low oncotic pressure as a cause of edema could be ruled out.

The owner requested a laparotomy, since the cow was valuable.

A laparorumenotomy was performed using a standart laparotomy incision in the left paralumbar fossa. Anesthesia of the paralumbar fossa and abdominal wall was

114 achieved by a proximal paravertebral nerve block. Prior to the rumenotomy an
115 abdominal exploratory was performed.
116 The ruminotomy was performed using a Weingarth's ring.
117 The exploratory revealed massive adhesions involving the spleen, reticulum, cranial
118 blind sac of the rumen, omasum and those parts of the liver that could be palpated
119 from the left flank incision. Two magnets with a 7.5-cm nail and loop of wire were
120 removed from the reticulum. An abscess, which was palpated on the caudomedial
121 wall of the reticulum, was lanced and drained into the reticulum and lavaged with an
122 iodine solution (Betadine®).
123 Postoperative treatment consisted of 10 litres of 0.9 % saline with 5 % glucose
124 administered intravenously daily, 1.2 x 10^6 IU/100 kg procaine penicillin (Procacillin®)
125 administered IM every eight hours and 1.1 mg/kg flunixin meglumine (Fluniximine®)
126 administered intravenously every 24 hours for 3 days.
127 Four days postoperatively, there was marked worsening of the brisket oedema (Fig
128 2) and development of mandibular oedema and swelling of the nose. In order to rule
129 out an allergic etiology of the swelling of the nose, the cow was given 2.5 mg
130 flumethasone (Cortival®) IV every 24 hours and 50 mg/100 kg tripelenamine
131 (Vetibenzamin®) IM once. Oedema of the front limbs and ventral abdomen (Fig 3)
132 developed seven days postoperatively. Ultrasonographic examination of the thorax
133 revealed severe pleural and pericardial effusion. Thoracocentesis yielded a
134 transudate with a specific gravity of 1.010 and no measurable protein.
135 Based on these findings, thrombosis of the cranial vena cava was suspected and the
136 cow was given 45,000 IU heparin IV every eight hours and 1 mg/kg furosemide
137 (Dimazon®) intravenously for three days. However, over the following three days, the
138 oedema worsened, breathing became laboured when the cow was recumbent, and

there was intermittent mouth breathing. Because of a poor prognosis and failure to respond to treatment, the cow was euthanased. A postmortem examination was carried out.

Postmortem examination confirmed the ultrasonographic diagnosis of massive adhesions involving the reticulum. A friable, beige, rough, 8 cm x 3 cm thrombus was fully occluding the lumen of the cranial vena cava (Fig 3). Histopathology of the thrombus revealed gram positive (Brown-Brenn staining) coccoid bacteria. Culture of the thrombus has not been performed.

The walls of the right atrium and ventricle of the heart were thicker than normal at 1 cm. The pericardial sac contained 100 ml of light red watery fluid. On cut surface, the pulmonary vessels contained several friable rough structures, up to 2 cm in length (Fig 4). The pulmonary parenchyma surrounding these areas was yellow. Culture of these lung lesions revealed *Streptococcus ssp*.

The hepatic bile ducts contained massive numbers of small liver flukes. Specimens of the lungs, kidneys and thrombus in the cranial vena cava were examined histologically. Based on the findings, the cow was diagnosed with thrombosis of the cranial vena cava, severe multifocal necrotising pneumonia with multiple pulmonary thrombi, ischemic renal infarction, *Dicrocoelium dentriticum* infestation and localised peritonitis in the region of the reticulum.

Discussion

Our patient had signs of thrombosis of the cranial vena cava. The differential diagnosis for sudden distension of both jugular veins includes obstruction of the cranial vena cava by a thrombus, compression of the vein by a mass and cardiac insufficiency. Because the cow had an elevated heart rate, echocardiography was

carried out and pericarditis, cardiomyopathy and endocarditis were ruled out. However, it was not possible to determine clinically whether there was obstruction or compression of the cranial vena cava. Radiography and ultrasonography showed no evidence of compression of the vein by a space-occupying lesion. Thus, obstruction of the cranial vena cava with a thrombus, which has been described in cattle (1,9,10), was diagnosed by exclusion of the other differential diagnoses.

In principle, thrombi, which result from increased coagulation or reduced blood flow, are differentiated from thrombi, which are attributable to suppurative inflammation. Wyssmann (1) described a thrombus, which was determined to be associated with a reticular abscess based on histological evaluation. In the present case, the thrombus causing distension of both jugular veins and impaired venous return with resultant oedema of the head and neck region was suspected to be septic based on histopathology.

The most common cause of thrombosis of the cranial vena cava is thrombophlebitis of the jugular vein (6). Septic emboli may also originate from other foci of infection, including mastitis, endometritis and claw disease (6,17). The most common pathogens are *Fusobacterium necrophorum* and *Actinomyces pyogenes*, but staphylococcus spp., streptococcus spp. and *Escherichia coli* may also be cultured from thrombi in the vena cava (18). No abnormalities were found on ultrasonographic examination of the jugular veins in our patient. The most likely source of infection was traumatic reticuloperitonitis with abscessation of the reticular wall. Peritonitis, mainly in the reticular region, in conjunction with thrombosis of the cranial vena cava was also described by Bueno (10) and Wyssmann (1). It is plausible that bacteria from the traumatic reticuloperitonitis were transported via the cranial epigastric and internal thoracic veins to the cranial vena cava.

The cow also had severe multifocal necrotising pneumonia with formation of multiple pulmonary thrombi. Metastatic bronchopneumonia caused by embolisation of part of a thrombus in the vena cava has been described (2,3,5,6,9). The clinical signs of thrombosis of the caudal vena cava and its sequelae are described as respiratory syndrome attributable to metastatic pneumonia, pulmonary thromboembolism or embolic pulmonary aneurysm (17). Selman (2) and Breeze (9) described respiratory syndrome due to thrombosis of the caudal vena cava and cranial vena cava, respectively. Pulmonary thrombi may lead to aneurysm and rupture of the vessel with subsequent epistaxis and sudden death. The lung lesions in the present case were consistent with thromboembolism. The most likely source for septic emboli traveling to the lungs was the septic thrombus in the cranial vena cava. A similar organism cultured from different lesions could prove that these lesions were related. Streptococcus ssp. could be cultured from the lung lesions, but no culture was performed from the thrombus in this case. However, histopathology of the thrombus revealed gram positive coccoid bacteria.

The clinical signs in our patient were attributable to oedema of the head and neck. Bueno (10) and Wyssmann (1) also described brisket and mandibular edema and bilateral jugular vein distension in cattle with thrombosis of the cranial vena cava. Oedema was not a clinical feature in the patient described by Breeze (9). However, all affected cattle had respiratory signs (1,9,10).

In human medicine, congestion of the cranial vena cava is usually the result of compression of the vessel, which in more than 80 per cent of patients is attributable to a malignant tumour (65% bronchial carcinoma). The lead sign in human patients is oedema of the head and neck (19). To the authors' knowledge, compression of the cranial vena cava causing congestion has not been reported in cattle.

Ultrasonography is an important diagnostic tool for assessment of the vena cava. Diagnosis of thrombosis of the vena cava directly by visualisation of the thrombus (7,20) or indirectly by identification of dilatation of the caudal vena cava in the region of the liver (5,21) is rare. A definitive diagnosis of thrombosis of the cranial vena cava can only be achieved by direct visualisation of the thrombus via ultrasonography. Bueno et al. (10) detected a thrombus visible within the right atrial lumen, extending into the cranial vena cava. However, we were not able to detect a thrombus in our patient. This was probably due to insufficient penetration into the thorax and lack of access to the cranial thorax because the examination area is delimited by the caudal margin of the thoracic limb. The cranial vena cava originates from the right atrium, traveling through the mediastinum to the thoracic inlet, where it bifurcates into the jugular veins. The cranial vena cava is covered by the lung lobes cranial to the heart. The vein can only be imaged adjacent to the heart.

Authors' contributions

Drs. Gerspach, Schweizer-Knubben, and Braun were the clinicians responsible for the case and described clinical and ultrasonographic findings and Dr. Wirz performed the necropsy and described the pathological findings.

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Figure legents

Figure 1
Physical appearance of the cow with brisket edema.

Figure 2
Physical appearance of the cow with distended jugular veins and brisket edema

Figure 3
Anatomical appearance of a friable, beige, rough, 8 cm x 3 cm thrombus from the cranial vena cava, bar = 1 cm.

Figure 4
Anatomical appearance of a thrombus within a pulmonary vessel, bar = 1 cm.